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Community-acquired methicillin-resistant *Staphylococcus aureus*: different populations, different results: reply from authors

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SIR, We are grateful to Sardana *et al.* for their interesting comments. They found, in contrast to the current published data and our results,[1,2] that community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) was not always responsible for severe skin manifestations. These different clinical presentations may be related to the different *S. aureus* clones involved in skin infections. It is becoming evident that the various CA-MRSA clones circulating in the different parts of the world harbour different antibiotic resistance profiles and toxin genes.[3] Therefore according to the clones circulating, the clinical manifestations may differ from one country to another. Most CA-MRSA clones harbour the Panton–Valentine toxin genes which are responsible for pus and necrosis and cause furuncles and abscesses, but other clones harbour exfoliative toxin genes responsible for blisters and outbreaks of impetigo.[4] Therefore it is important to identify the circulating CA-MRSA clones in a geographical focus, better to characterize the clinical manifestations associated with these clones.

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A surgical treatment for anxiety-triggered palmar hyperhidrosis is not unlike treating tearfulness in major depression by severing the nerves to the lacrimal glands

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SIR, The recent study of Krogstad *et al.*[1] in this Journal, reporting on the daily pattern of sweating and response to stress in patients with palmar hyperhidrosis, is an important contribution to the clinical literature. Patients and controls did not differ while doing 'physical activity' or 'other activity'. In contrast, in patients, but not in controls, palmar hyperhidrosis was most severe at mid-day during the working day (when psychological stress levels are likely to be highest). These authors conclude that their 'finding supports the idea that thermoregulatory sweating is normal in hyperhidrotic patients' but found extensive evidence that psychological stress influences patients with palmar hyperhidrosis more than controls.[1]

The surgical literature (see Krogstad *et al.* for references) advocates sympathectomy or other surgical procedures as the first line of treatment for palmar hyperhidrosis. This is despite the frequent postsympathectomy complications, most commonly truncal compensatory hyperhidrosis, a condition which we contend is at least as uncomfortable and as socially embarrassing as palmar hyperhidrosis. Long-lasting postsurgical compensatory truncal hyperhidrosis has been reported to occur in 44–86% of patients.[2,3]

Palmar hyperhidrosis of clinical severity is a hallmark physical sign of many anxiety disorders, including generalized anxiety disorder, panic disorder, posttraumatic stress disorder, and especially social phobia.[4] These are increasingly well understood and highly treatable neurobiological conditions. They are moderately heritable hard-wired fear responses,[5] and are linked to amygdalar and locus coeruleus hyper-reactivity during psychosocial stress.[6,7] Anxiety disorders are known to be much more common among women. This is consistent with the finding of Krogstad *et al.* that among controls sweating was reported more often by men, while among the hyperhidrosis group sweating was reported more often among women.

It may be relevant that a disproportionate percentage of the published surgical studies of palmar hyperhidrosis has been

conducted in Asian countries where a strong stigma exists against having any mental disorder. Because of this, many individuals from these cultures (e.g. Japan, Korea, Taiwan and Hawaii) would rather go under the knife than take psychotropic medications. However, all patients with palmar hyperhidrosis should be offered and strongly encouraged to complete a full course of a psychopharmacological treatment (e.g. with a serotonin/noradrenaline reuptake inhibitor) for anxiety, the underlying cause of palmar hyperhidrosis,[4,8] before undergoing a surgical procedure. A surgical treatment for anxiety-triggered palmar hyperhidrosis is not unlike treating tearfulness in major depression by severing the nerves to the lacrimal glands. We have recently made a similar argument advocating a psychopharmacological, rather then a surgical, first-line treatment for blushing.[9]

Finally, it cannot be more strongly emphasized that, like major depression, the above anxiety disorders are a risk factor for suicide.[4] Referring an anxious patient with palmar hyperhidrosis to surgery without first completing a proper trial of psychotropic medication may constitute malpractice especially if the patient experiences some of the more severe surgical complications which can occur during sympathectomy.

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Immotile acral melanocytes?

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Sir, Eccrine poroma (EP) is a frequently encountered neoplasm which usually presents as a flesh-coloured nodule on the soles of the feet and palms of the hands. We have previously reported a pigmented facial EP.[1] We recently encountered two more patients with pigmented EP located on the abdomen and buttock, respectively. We are intrigued by the locations of the pigmented variant of this adnexal tumour. As this tumour appears predominantly on the distal extremities, it is surprising that very few reports have documented pigmented variants of EP occurring at these sites. In the biopsy specimens of pigmented EP, dendritic melanocytes (MCs) were usually seen among the tumour lobules. Several hypotheses have been proposed to explain the mechanism of MC colonization in the pigmented tumours. It is known that sweat duct primordia contain MCs during the 14th week of gestation that are lost later in embryonic development.[2] Therefore, it is possible that certain tumour-related factors may activate MCs in the sweat gland acrosyringium after fetal life. This hypothesis, however, fails to explain the scarcity of reports on acral pigmented EP. Another explanation is that the MCs in the epidermis surrounding the tumour might have migrated to the tumour nest during tumour proliferation via the secretion of tumour-related factors such as fibroblast growth factor or endothelin-1.[1,3] This proposed mechanism may provide a better explanation for the observed clinical phenomenon. Interestingly, in clinical situations involv-